REMARKS

Claims 12-107 are all the claims pending in the application.

New claims 12-43 recite a composition comprising an N-linked high mannose type oligosaccharide derived from the major outer membrane protein of Chlamydia, or a structurally identical oligosaccharide, or a derivative of the oligosaccharide; wherein the N-linked high mannose type oligosaccharide comprises at least five mannose residues, comprises a trimannosyl core consisting of at least two mannosyl branches, one branch linked $\alpha 1 \rightarrow 6$ and the other branch linked $\alpha 1 \rightarrow 3$ to a mannose residue and having mannosyl substitution or branching at the $\alpha 1 \rightarrow 6$Man residue of the trimannosyl core and mannosyl substitution or branching at the $\alpha 1 \rightarrow 3$Man residue of the trimannosyl core; wherein said N-linked high mannose type oligosaccharide is capable of binding to host cells to thereby inhibit binding of Chlamydia to said host cells, and wherein said N-linked high mannose type oligosaccharide is present in the composition in an amount sufficient to inhibit binding of Chlamydia to said host cells; and a biologically acceptable carrier, diluent or excipient.

Claims 44-75 recite an isolated N-linked high mannose type oligosaccharide derived from the major outer membrane protein of Chlamydia, or a structurally identical oligosaccharide, or a derivative of the oligosaccharide, wherein the N-linked high mannose type oligosaccharide is capable of binding to host cells to thereby inhibit binding of Chlamydia to said host cells.

Claims 76-107 recite the multivalent oligosaccharide derived from the major outer membrane protein of Chlamydia, or a structurally identical oligosaccharide, or a derivative of the multivalent oligosaccharide, linked to a carrier wherein the multivalent oligosaccharide is capable of binding to host cells to thereby inhibit binding of Chlamydia to said host cells.

Support for the recitation that the N-linked high mannose oligosaccharides are derived from the outer membrane protein of Chlamydia and are capable of binding to host cells to thereby inhibit binding of Chlamydia to said host cells is found throughout the specification, and particularly in the examples. Support for the structure of the oligosaccharides is found in the original claims and throughout the specification, particularly from pages 9-11 and in tables 1 and 2. Accordingly, no question of new matter arises and entry of this Amendment are respectfully requested.

In the final Office Action in the parent application, the then pending claims were rejected under 35 U.S.C. § 102(b) as being anticipated by *Swanson et al.* (U). The Examiner asserted that the glycan identified by *Swanson et al.* must necessarily be the same as the carbohydrate recited in the rejected claims, because the method *Swanson et al.* uses to separate the glycan of the major outer protein (MOMP) of *Chlamydia trachomatis* is the same as that taught in the present application. The Examiner further asserted that *Swanson et al.* concludes that the experimental results suggest that the glycan portion of the MOMP of *Chlamydia trachomatis* is involved in the attachment process in HeLa cells and that the publication exemplifies inhibiting binding of Chlamydia to a mammalian cell.

For the following reasons, the new claims distinguish over *Swanson et al.*

The present application is based on the following discoveries:

(1) That, surprisingly, a high mannose-type oligosaccharide is linked to the major outer membrane protein (MOMP) of Chlamydia; and

(2) That, based on the fact that high mannose-type oligosaccharide inhibits Chlamydia infection, surprisingly, this high mannose-type oligosaccharide is the site that binds to host HeLa cells leading to infection.

The definition of the high mannose-type oligosaccharide and its structure are not the basis of patentability. Rather, the crucial point is the unique occurrence of the high mannose-type oligosaccharide at the MOMP of Chlamydia, the ability of the high mannose-type oligosaccharide of MOMP to directly bind to the host cell leading to infection, and the ability of the high mannose-type oligosaccharide to inhibit infection.

While occurrence of N-linked oligosaccharides in some bacteria has been known, although this is rare, high mannose-type oligosaccharides from bacterial sources was not known prior to the present invention.

The present claims require that the oligosaccharide, or a structurally identical oligosaccharide or derivative be capable of binding to host cells to thereby inhibit binding of Chlamydia to the host cells. However, *Swanson et al.* teach that because the glycan's conformation might not be the same as that of the intact glycoprotein, the interaction between major outer membrane protein in the EB and the HeLa cells may differ from the interaction between the glycan and the HeLa cells. (*Swanson et al.*, page 27, right column, first full paragraph.) Thus, *Swanson et al.* do not teach a specific utility for the structure they isolate and describe. Accordingly, *Swanson et al.* cannot teach the claimed composition or oligosaccharides which, as claimed, must be capable of inhibiting binding of Chlamydia to host cells. *Swanson et al.* is no more than an invitation to experiment.

Furthermore, as to the structure of the oligosaccharide, *Swanson et al.* do not teach how the structure of the oligosaccharide can be determined. Identifying a moiety as a "glycan" or as an "oligosaccharide" is very different from describing a specific structure which is an infection site and which inhibits infection. This is especially true of the present invention where the claims recite that the oligosaccharide is a N-linked glycoprotein not

heretofore known to be present in bacteria or other microorganisms. There are hundreds of different glycoproteins, and the fraction isolated by *Swanson et al.* could have been any of these hundreds of glycoproteins. In fact, even *Swanson et al.* did not know what they had isolated. *Swanson et al.* teach that the structure may contain galactose (see Table 1). In the experiment which looked to the effects of sugars on binding of Chlamydia glycan to HeLa cells, competition assays were performed using three aldoses: mannose, galactose and N-acetylglucosamine to block the binding of glycan. The experiments showed that all three sugars inhibited the binding of glycan to HeLa cells, while two ketoses did not. Yet the claimed structure does not contain galactose, as hypothesized by *Swanson, et al.*

Accordingly, it is believed that the Examiner's rejection has been made in hindsight. That is, knowing what the specific structure of the carbohydrate is, the Examiner is now saying that the specific structure is taught by the reference, even though *Swanson et al.* does not identify the glycan in terms of its structure, except to say that it may contain galactose, which the presently claimed glycan does not, and even though glycans can have hundreds of different structures.

Lack of novelty, *i.e.,* anticipation, requires that the same invention, including each element and limitation of the claims, was known or used by others before it was invented by the patentee. Proof of anticipation requires prior knowledge by others that the claimed subject matter is expressly or inherently described in a single prior art reference. The single reference must describe and enable the claimed invention, including all claim limitations, with sufficient clarity and detail to establish that the subject matter already existed in the prior art and that its existence was recognized by persons of ordinary skill in the field of the invention.

In view of the above, the new claims are patenable over *Swanson et al.* and allowance thereof is requested, respectfully.

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